PSYCHIATRIC SEQUELAE IN HEAD INJURY PATIENTS – A case control study

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BRANCH – XVIII

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CERTIFICATE

This is to certify that the dissertation entitled "**PSYCHIATRIC SEQUELAE IN HEAD INJURY PATIENTS – A case control study"** is the bonafide original work of **Dr. P. Poorna Chandrika.** in partial fulfillment of the requirements for **M.D. (Psychiatry) BRANCH – XVIII** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in March 2007.

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DECLARATION

I, **Dr. P. POORNA CHANDRIKA** solemnly declare that dissertation titled, "**PSYCHIATRIC SEQUELAE IN HEAD INJURY PATIENTS – A case control study"** is a bonafide work done by me at Stanley Medical College during 2004-2007 under the guidance and supervision of **Dr. Prof. M. Thirunavukarsu, M.D., D.P.M.** Professor and Head, Department of Psychiatry, Stanely Medical College, Chennai-600 001.

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CONTENTS

INTRODUCTION

CLASSIFICATION OF HEAD INJURIES

The head injuries are classified according to severity of head injures. The severity of head injures is best gauged by the depth and the duration of the impairment of consciousness following the head injury. In gauging the head injury. Glasgow Coma Scale (GCS) is most commonly used (Teasedale & Jennet).

Head Injury Classification

Head Injury	Glasgow Coma Scale
Mild	13-15
Moderate	9-12
Severe	3 -8

Head injuries can also be classified as :

1) Penetrating
2) Non penetrating

According to nature of damage as :

- Concussion
- Contusion
- Laceration

According to site of Injury

- Right
- Left

- Bilateral
- Frontal
- Parietal
- Temporal
- Occipital
- Frontoparietal
- Parieto occipital
- Temporoparietal

According to site of bleed

- Extradural
- Epidural
- Subdural
- Subarachnoid
- Intracerebral

Head injury is 2nd most important causes of mortality after cancer. Early mortality has considerably improved as a result of advances in the management of the early acute stages.

Chronic sequale remain, a challenge to medical care and communal resources, mental sequale outstrip the physical as a cause of difficulty with rehabilitation, hardship at work, and social incapacity generally.

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CLINICAL FEATURES

Acute Behavioral consequences.

Most patients admitted to hospital have a mild injury. Minority of these patients develop acute complications . brain swelling, delayed haematoma and intracranial infection) or prolonged postconcussional symptoms.

Most common consequence of head injury is impairment of consciousness, ranging from transient confusion to protracted coma. The Glasgow coma score (GCS) is commonly used to grade the severity of traumatic brain injury. Scale gives a quantitative assessment of level of consciousness and neuro-logical status based on patterns of eye opening, as well as best verbal and motor responses.

GCS scores between 13 and 15 define mild brain injury, 9-12 define moderate brain injury and scores between 3 and 8 define severe brain injury.

Early phase of recovery from traumatic brain injury is characterized by disorientation, confusion and impaired memory function. Post traumatic amnesia occurs during the period when the patient is disoriented and confused. Duration of post traumatic amnesia used as a measure of traumatic Brain Injury.

Duration of post traumatic amnesia has proved to be a good predictor of the degree of disability, vocational outcome and severity of personality change after traumatic brain injury. Delirium in brain injury patients may be due to structural brain damage, cerebral edema, brain hypoxia, seizures, electrolyte imbalance, infections, medications or alcohol withdrawal.

Chronic Behavioral Consequences

Cognitive Disorders – Cognitive disturbances are one of the most important long term sequale of severe traumatic brain injury. Study on 127 patients after 1 year follow up showed slower information processing and greater impairment in memory than control group. Memory functions are also impaired in traumatic brain injury patients. DSM IV T.R diagnosis of amnesia disorder due to traumatic brain injury and chronic subtype may be made for those non demented patients.

Several neuro psychological tasks are present to quantify these deficits.

DEMENTIA

Dementia is a syndrome defined by DSM – IV TR by impairment of memory and at least one other cognitive domain in the absence of alteration of consciousness.

Apart from memory disturbances, these patients may be severely apathetic and withdrawn.

Traumatic brain Injury is associated with expression of amyloid precursor protein, oxidative stress and an increased deposition of amyloid – B peptides that leads to onset of dementia.

PERSONALITY CHANGES

Traumatic Brain Injury patients may experience significant personality changes. These patients may be irritable, childish, anxious or aggressive. Disinhibition is frequent and striking clinical feature that may lead to antisocial behavior, at the other end they may become apathetic, abulic and withdrawn.

Some investigators divide these changes in to 2 syndromes a pseudo-depressed personality syndrome characterized by apathy and blunted affect, and a pseudopsychopathic personality syndrome which is characterized by disinhibition, egocentricity and sexual ina propriateness. DSM – IV TR defines personality change due to traumatic brain injury as a persistent personality disturbance that represents a change from individual's previous personality.

Roberts (1976, 79) survey of long term outcome of severe head injuries found the commonest pattern of personality change had a distinctly frontal character, termed it as "fronto – limbic dementia".

Temporal lobe injury associated with aggression and poor impulse control.

MOOD DISORDERS

DEPRESSIVE DISORDERS

Depressive disorders appear to be frequent complication among patients with traumatic brain injury patients.

According to DSM – IV TR diagnostic criteria, depressive disorder due to brain injury is divided in to

(1) with major depressive like episode.

(2) with depressive features.

Frequency varies from 6 to 77 percent, in another study major depression frequency was 61 percent. In another study 2 years after injury depression was found to be 42 percent.

In another study 91 patients followed up for 2 years. 47 patients developed mood disorder in the first year of follow up. Major depressive disorder was occurred in 30 patients, nine patients had minor depression and remaining had manic or mixed episodes. Concluded that approximately one half of traumatic brain injury patients develop mood disorder in the first year of injury.

Patients who were vulnerable developing mood disorders were not significantly different with regard to demographic variables, severity of brain injury or degree of functional disability.

Depressed patients were more likely to have a personal history of mood and anxiety disorders than the nondepressed group.

Analysis of the oxford collection of head injury Records suggested that depressive symptoms were more common among patients with right hemisphere lesions. Symptoms of depression also more among patients frontal and parietal lesions than among patients with other lesion locations.

(Jorge, Robinson) study of depressive disorder in patients after traumatic Brain Injury was associated with left dorsolateral frontal or left basal ganglia lesions.

Most consistent clinical correlate of depressive disorder is poor psychosocial adjustment.

Secondary Mania – have been reported in a number of organic disorders such as thyroid disease, uremia and vitamin B12 deficiency or after open heart sugery.

Recent study reported 6 of 66 patients had secondary mania one year after injury. Secondary mania was not related to type or severity of

brain injury but a association was found with lesions in the ventral and anterior aspects of temporal lobe.

Differential diagnosis of mania after traumatic brain injury include substance induced mood disorder, psychotic Syndrome associated with epilepsy.

ANXIETY DISORDERS

Anxiety disorders after traumatic brain injury may manifest themselves as pathological worrying, anxious foreboding and autonomic symptoms.

Prevalence of obsessive-compulsive disorder occurring after traumatic brain injury has been estimated to be 2 to 4 percent.

Post Traumatic Stress Disorder following TBI is characterized by recurrent intrusive recollections, distressing dreams, and flash backs of traumatic event.

There has been controversy regarding whether PTSD can develop after traumatic brain injury, it has been thought that loss of consciousness is inversely proportional to development of PTSD

Prevalence of PTSD to be 27 to 38 percent after traumatic brain injury.

Vulnerability to anxiety disorders after traumatic brain injury has been studied extensively but no specific characteristic has been noted in

terms of severity of brain injury, or social, physical or cognitive impairment but one study noted the symptoms present with right orbito frontal cortex lesions.

In patients with anxious depression focal right hemispheric lesions was found.

PSYCHOTIC DISORDERS

Frequency of psychotic disorders in Traumatic brain injury was 7%, these symptoms were observed within 2 years after head injury positive symptoms were more frequent.

PATHOLOGY OF BRAIN INJURY

- Neuropathological Classification of Traumatic Brain Injury lesions.

FOCAL LESIONS

- Intra cranial – extra cerebral haemorrhage (epidural and subdural hemotomas and subarachnoid hemorrhage).
- Intra cerebral hemorrhage.
- Focal ischaemic lesions.

DIFFUSE LESIONS

- Diffuse axonal injury.
- Diffuse ischaemic damage.

PATHOLOGY

Types of primary and secondary brain injuries.

First level of categorization of head injury divides them into closed or penetrating injuries depending on the integrity of meningeal coverings. Missile wounds are the most frequent cause of penetrating brain injuries.

Motor vehicle accidents are the most frequent cause of closed trauma, which represents the majority of TBI.

Primary Brain damage is produced by contact and inertial forces that occur at the time of injury. Contact forces may result in laceration to the scalp, skull fractures, intracranial hemorrhages, contusion and intracerebral hemorrhages.

Inertial loading consists of acceleration, deceleration and rotational forces that result in diffuse axonal injury and eventually acute subdural hematoma from the tearing of subdural bridging veins.

Secondary brain damage is produced by pathological processes that are initiated at the moment of injury but spans a variable period after the traumatic episode.

These include brain damage secondary to ischaemia (that resulting from hypertension or hypoxia or both), brain swelling, raised intracranial pressure and infection.

Focal lesions consists of contusions and lacerations that usually occur at the surface of brain. More prominent at the crest of cerebral gyri and have a predilection for the frontal and temporal poles. Lacerations are usually accompanied by extracerebral hemorrhages (burst lobe).

Intracranial extracerebral hemorrhages occur in the epidural space. (ie between the skull and dura), the subdural space (ie between dura and arachnodi) or the subarachnoid space.

Intracerebral hemorrhages are often multiple involving frontal and temporal lobes and basal ganglia and may have a delayed onset (ie hours or days after trauma).

Diffuse lesions include diffuse axonal injury which occur within corpus callosum, thalamus and dorsolateral quadrants of the upper brainstem. Pathologic processes include fragmentation of the axolemma, axonal transport disruption axonal bulb formation, astrogliosis and microglial activation.

Ischaemic damage is highly prevalent among patients with head injuries.

The influence of these different patterns of injury on the psychiatric disorders after traumatic brain injury has not been extensively studied.

Focal and diffuse lesions usually coexist in Traumatic brain injured patients. .

COGNITIVE IMPAIRMENT AFTER HEAD INJURY

Cognitive impairment is, of course, the direct result of the damage to brain tissue which has occurred. Minor injuries are compatible with full intellectual recovery, even when indubitable loss of consciousness has occurred, in the sense that the patient feels himself to be unimpaired and psychometric tests

Symptoms and symptom groups seen 1-5 years after penetrating injury
in relation to location of brain damage
(from Lishman, 1968)
(indicates strong evidence of special association)

	Left	**Right**	**Frontal**	**Parietal**	**Temp**	**Occipital**
Intellectual Disorder	+			+	+	7+
General Intellectual impairment	+				+	7+
Dysphasia	+			+	+	
Impairment of Memory	+			+		+
Difficulty I concentration		+				+
Depression		+	+			
Anxiety						+
Irritability		+	+	+		
Aggression			+			
Euphoria			+			
Behavioural Disorder		+	+			
Headache or dizziness			+			
Fatigue				+		
Sensitivity to noise			+			

Treatment of head injury :

The treatment of acute stages of head injury and neurosurgical interventions and early complications have not been covered up as they are rarely dealt by the psychiatrists.

Once the patient starts recovering and is out of neurosurgical care psychological management has great importance in determining the prognosis of the patient.

Early Management :

Initial convalescent period is usually undertaken in hospital and ideally in an atmosphere as free from stress as possible. Mental exertion should be avoided at the same time physical activity should be encouraged. Gradual exercises should be started with would also boost up patients morale.

Time should be devoted to exploration of patient's anxieties. Fears should be brushed aside. Explanations should be given regarding the initial symptoms. More detailed and specialized care is required by patients who have more severe brain damage with neurological sequelae and intellectual impairment. Also special attention should be given towards patients with minor injures but with persistent psychiatric disability.

Neurological Sequelae

These are treated mainly with the help of neurologist, physiotherapist and occupation therapist.

Rehabilitation of cognitive functions is a very challenging task. Full psychometric assessment is very essential. There may be impairments of memory, verbal ability, comprehension, Visuospatial ability, attention and manual dexterity.

A optimistic approach is required in order to install enthusiasm with ready allowance for fatigue and tolerance for short comings. The programme should be graded with goals at any stage which should rational, realistic and achievable.

Xangwill has stressed that the emphasis should be placed on reeducation, compensation and substitution in rehabilitation.

Reeducation involves retraining of the patients in skills and accomplishments which have been impaired. Compensatory functions must be trained, if attempts at reeducation fail. Compensatory functions like using props for memory or methods of expression in severe motor dysphasia can be used. Substitution is required when damage to a particular function is irreparable. Newcombe had reviewed various strategies to assist rehabilitation with cognitive retraining.

Personality changes following head injury are difficult to modify. Psychotherapy is relatively superficial and should be aimed to help the patient to achieve some insight into what he is lacking. A behavioural modification should be tried to reduce disruptive behaviour and encourage constructive behaviour. Psychotherapy and hebavioural therapies may be of use in patients with depression, phobias or headaches and postconcussional syndrome.

Tranquillizing drugs such as benzodiazepaines may help to relieve tension and anxiety. Antidepressants will help in depression. Chlorpromazine will be of help in patients with frontal lobe injures. Antipsychotic will help in acute delirium, psychosis and outburst of violence. Anticonvulsants like carbamazapine may be of help in preventing outburst of aggression.

REVIEW OF LITERATURE

HISTORY :

Although the psychological changes arising after had injures have been known since ages, the case of Phineas Gage in 1848 is the first detailed clinical report of pathological personality change after head injury. While Gage was working with explosives a pointed iron rod blasted through his left frontal lobe and temporal pole. Gage survived for eleven years but his personality was not the same. From well balanced, honest, reliable person he was transformed into childish inconsiderate person lacking judgment. In 1888, Leonore Welt published a similar case, after death, the autopsy of this patient showed lesion in right inferior frontal gyrus. After this multiple cases have come up not only showing personality changes but also other psychiatric changes following head injury.

Rapoport MJ et al. 2006. Patients with TBI had poorer processing speed, verbal memory, language, and executive function; they self-reported more psychologic distress, psychosocial dysfunction, and postconcussive symptoms; and they were rated as more impaired in functioning than the comparison group. TBI of moderate severity accounted for most of the between-group differences. TBI, particularly of moderate severity, led to poorer cognitive and psychosocial

functioning one year postinjury among older adults. The clinical significance of this may become more evident with time in this vulnerable population.

Koponen S et al 2006. One-third of the subjects had traumatic lesions visible on MRI. Only three psychiatric disorders, that is, delusional disorder, dementia, and the disinhibited type of organic personality syndrome, were significantly more common in subjects with contusions. Concerning the location of contusions, organic personality syndrome and its disinhibited subtype were associated with frontal lesions, and major depression was, surprisingly, inversely associated with temporal lesions. These results, which should be interpreted with caution due to the limited size of the study group, suggest that the majority of psychiatric disorders after traumatic brain injury are not closely related to the specific location or even the presence of contusions detectable with post-acute MRI.

Jorge RE, et al. 2005. Mood disorders, particularly major depression, are the most frequent complication of traumatic brain injury. Major depression is present in about 40% of patients hospitalization for a traumatic brain injury. Anxiety disorders, substance abuse, dysregulation of emotional expression, and aggressive outbursts are frequently associated with major depression, and their

coexistence constitutes a marker of a more disabling clinical course. The complex interactions of genetic, developmental, and psychosocial factors determine patients' vulnerability to developing affective disturbances following a traumatic brain injury.

Glaesser J et al. 2004. 27% of the sub-sample who were not unconscious for an extended period but only 3% (1 of 31 patients) who were unconscious for more than 12 hours as a result of the accident were diagnosed as having current PTSD ($P < .02$). TBI and PTSD are not mutually exclusive. However, victims of accidents are unlikely to develop a PTSD if the impact to the head had resulted in an extended period of unconsciousness.

Slewa-Younan S et al. 2004. No significant sex differences existed in the outcome measures or in injuries not associated with the central nervous system. Few investigations exist on the effect of patient sex on measures of injury severity and outcome after a TBI. In the present study, men's levels of injury severity were greater than women's despite the same admission criteria (high-speed MVC) being applied to both sexes.

Jorge RE, et al. 2004. Prospective, case-controlled, surveillance study conducted during the first year after the traumatic episode occurred. Major depressive disorder was observed in 30 (33%) of 91

patients during the first year after sustaining a TBI. Major depressive disorder was significantly more frequent among patients with TBI than among the controls. Patients with TBI who had major depression were more likely to have a personal history of mood and anxiety disorders than patients who did not have major depression. Patients with major depression exhibited comorbid anxiety (76.7%) and aggressive behavior (56.7%). Patients with major depression had significantly greater impairment in executive functions than their nondepressed counterparts. Major depression was also associated with poorer social functioning at the 6-and 12-month follow-up, as well as significantly reduced left prefrontal gray matter volumes, particularly in the ventrolateral and dorsolateral regions.

Jorge R, et al. 2003. Mood disorders are a frequent complication of traumatic brain injury that exerts a deleterious effect on the recovery process and sychosocial outcome of brain injured patients. Prior psychiatric history and impaired social support have been consistently reported as risk factors for developing mood disorders after traumatic brain injury (TBI). In addition, biological factors such as the involvement of the prefrontal cortex and probably other limbic and paralimbic structures may play a significant role in the complex pathophysiology of these disorders. Mood disorders occurring after TBI

are clearly an area ofneuropsychiatry in which further research in etiology as well as treatment is needed.

Tateno A et al. 2003 Aggressive behavior was significantly associated with the presence of major depression, frontal lobe lesions, poor premorbid social functioning, and a history of alcohol and substance abuse.

Andersson S et al. 2002. Apathy is associated with specific cognitive deficits related to frontal lobe dysfunction. The results are in accordance with the definition of apathy and confirm apathy-cognitive function relationships reported in other neurologic populations.

Starkstein SE et al. 2002. The best approach is to assess the presence of depressive symptoms using semi-structured or structured psychiatric interviews such as the Present State Exam, the Structured Clinical Interview for DSM-IV, or the Schedules for Clinical Assessment in Neuropsychiatry. The diagnosis of a depressive syndrome should be made using standardized diagnostic criteria for mood disorders due to neurological disease such as in the DSM-IV or the ICD-10. Depression rating scales, such as the Hamilton Depression Scale and the Center for Epidemiologic Scales for Depression may be used to rate the severity of depression and monitor the progression of antidepressant treatment.

Deb S, et al. 1999. In comparison with the general population, a higher proportion of adult patients had developed psychiatric illnesses 1 year after a traumatic brain injury; the rates of depressive episode and panic disorder were significantly higher in the study group. A history of psychiatric illness, an unfavorable global outcome according to the Glasgow Outcome Scale, a lower score on the Mini-Mental State examination, and fewer years of formal education seemed to be important risk factors in the development of a psychiatric illness.

McGuire LM. Et al. 1998. Multiple injuries were most common in psychiatric patients. The role of TBI in the emergence, expression and treatment outcome of psychiatric disorders and the risk factors that leave psychiatric patients vulnerable to TBI should be further examined.

Fann JR. et al. 1995. Depression and anxiety are common in outpatients with traumatic brain injuries. Patients with depression or anxiety are more functionally disabled and perceive their injury and cognitive impairment as more severe. Depressed patients report more increasingly severe postconcussion symptoms.

Morton MV et al 1995. The majority of individuals who sustain TBI are young males between the ages of 19 and 25, who are in the early stages of establishing their independence in areas including friendships, leisure activities, intimate relationships, residence, and

employment. Depicts that individuals who experience severe TBI are at high risk for a significant decrease in their friendships and social support relates to the lack of opportunity for establishing new social contacts and friends. relates to the decrease in leisure activities for individuals with severe TBI. Finally, anxiety and depression are found at high levels for prolonged periods of time following severe TBI. Several clinical recommendations are drawn from this literature review. They are: (1) Clinicians such as psychiatric social workers, psychologists, or psychiatrists may need to be called upon more quickly for intervention. The treating physiatrist cannot be expected to provide the insight and frequency of psychological services that may be necessary for many of these patients. (2) Since the literature seems to suggest that the presence of one psychosocial deficit, e.g., anxiety, will often be followed by other similar types of problems, e.g. depression, the treatment team needs to be sensitive to how rapidly these problems can cascade into a very dangerous situation. (3) Perhaps the most compelling recommendation we can make is for community rehabilitation specialists to focus significantly more energies and resources upon the psychological health of clients with TBI. Staff need to be trained to detect these signals that clients with TBI are often sending. It is apparent that psychosocial factors contribute to a rising obstacle level to community adjustment.

Fedoroff JP et al 1992. The presence of left dorsolateral frontal lesions and/or left basal ganglia lesions and, to a lesser extent, parietal-occipital and right hemisphere lesions was associated with an increased probability of developing major depression. Compared to the nondepressed group, the group with major depression had a higher frequency of previous psychiatric disorder and showed evidence of poorer social functioning. Major depression occurs in about one-quarter of patients after traumatic brain injury. This is the same frequency as in other major disorders such as stroke. Major depression appears to be provoked by one or more factors that include poor premorbid social functioning and previous psychiatric disorder or injury to certain critical brain locations.

The main finding of the present study was the high rate of most axis I and II disorders during the 30 years after traumatic brain injury. These findings suggest that traumatic brain injury not only temporarily disturbs brain function but may cause decades-long or even permanent vulnerability to psychiatric disorders in some individuals.

The rates of lifetime and current psychotic disorders were both 8.3% (95% CI=2.8%–18.4%). They were significantly higher than the prevalences in the ECA survey

(1.5% and 0.7%, respectively) (25). Five percent of our patients (95% CI=1.0%–13.9%) had delusional disorder, whereas the estimate for its population prevalence is only 0.03% in DSM-IV. Our figures are in accordance with the rate of 9% for psychoses (2% for paranoid psychoses) after traumatic brain injury found in an early study by Achte et al.

All eight patients with very severe cognitive impairment were male. This finding cannot be explained by alcohol use in men, because the presence of very severe cognitive impairment according to the Mild Deterioration Battery was not associated with lifetime alcohol abuse or dependence (Fisher's exact test, p=1.00). Nor were there significant differences in the severity of brain injury between men and women (Fisher's exact test, p=0.24). The neuroprotective influences of estrogen and progesterone (28) can, at least partially, explain this more favorable outcome after traumatic brain injury in women.

In population studies (29–31) the total prevalence of personality disorders has ranged from 5.9% to 13.5%. Also, Hibbard et al. (11) reported that avoidant (26%) and paranoid (26%) personality disorders are common after traumatic brain injury. It seems that traumatic brain injury can expose some individuals to social anxiety, suspiciousness, and detachment. There were only two patients (3.3%) with definite

cluster B personality disorders in our group. This is in contrast to the findings of Hibbard et al. (11), who found borderline personality disorder to be the most prevalent disorder after traumatic rain injury (34%).

Robert van Reekum et al. There are now a number of studies examining the issues related to DSM- or ICD-based psychiatric disorders after TBI. Although these studies have methodological limitations as discussed above, there is a strong and growing body of evidence to support the hypothesis that TBI frequently causes some, but not all, psychiatric disorders in those who have suffered a TBI. There is compelling evidence of causation for *major depression*, *bipolar affective disorder*, and the *anxiety disorders* after TBI. The evidence for psychosis and substance abuse suggests that TBI imposes either no increased risk or a very minor increased risk of these disorders.

These results strongly support the need for a thorough and reliable assessment of mood, anxiety, and personality disorders in all persons who have suffered a TBI. Psychiatric illness, even in the absence of TBI, can cause impairment and disability contributing to handicap.

In terms of the prevalence of psychiatric disorders, major depression was the most common, at approximately 44% across all

available studies. Bipolar illness was much less frequent, at approximately 4%. The anxiety disorders were common, ranging from approximately 6.5% for OCD to a high of approximately 14% for PTSD. Substance abuse was also fairly common, at 22%, while psychosis was uncommon at less than 1%.

In terms of the *temporal sequence*, it is clear from the data that some psychiatric disorders were present in some patients prior to the TBI. However, it is also clear that many more subjects had apparent onset of their disorders after the TBI.

In terms of *biologic plausibility*, there has been considerable research related to the mood disorders after TBI, but much less so for the other psychiatric disorders.

Daryl Fujii et al. 2000. Reports vary as to the severity of TBI required to trigger a psychosis. Some studies suggest that individuals who develop a psychosis after TBI had generally sustained moderate to severe head injuries. By contrast, many case studies report development of a psychosis after mild brain injuries with no loss of consciousness

Determining the etiological significance of TBI in the development of psychosis is the latency between the injury and onset of symptoms. One study reported latencies ranging from 2 days to 48 years.[4] Another reported latencies between 3 months to 19 years.

Preliminary evidence reports a preponderance of temporal lobe abnormalities on CT/MRI and EEG. Frontal lobe abnormalities on CT/MRI are also common but are less consistently found.

Difficulty in diagnosis of PDTBI is ruling out other mental disorders. Many schizophrenics sustain TBI during their lifetime.

Males are at higher risk for developing PDTBI. Males are reported to have a higher incidence of other neurodevelopmental disorders such as learning disabilities.

Delusions appear to be more common than hallucinations. The most common delusion is the persecutory type, most common hallucination was auditory. Patients with later onset of symptoms (2 years or longer after the trauma) were more likely to have hallucinations than early-onset patients (onset less than 2 years after trauma).

The course of illness for a significant majority of subjects in our analysis was good. Prognosis did not appear to be affected by variables such as gender, delay in onset of psychosis after TBI, or diagnosis of a seizure disorder.

Results are similar to other findings that the majority of patients who develop a PDTBI sustained a moderate to severe head injury. Still, in all of these studies there were patients who developed PDTBI after a mild TBI, including some who did not lose consciousness.

Results are very similar to those in a previous meta-analysis of the literature, which reported that most closed head injury patients who demonstrate psychotic symptoms do so within the first year (52%) and the majority within 5 years (85%), while the majority of moderate to severe TBI patients who report psychotic symptoms do so within the first year (67%).

About 65% of the cases reported positive findings on MRI/CT. The most common location of findings was the frontal lobes, followed by the temporal lobes and ventricles.

First, PDTBI patients appear less likely to demonstrate negative symptoms than schizophrenic patients. In our sample, only 14% reported the presence of negative symptoms, whereas the prevalence rate of negative symptoms in schizophrenia has been reported to range from 25% to 84%.

On MRI/CT, a higher percentage of PDTBI than schizophrenic patients demonstrate positive findings. For PDTBI patients, there appeared to be an equal distribution of focal signs (62%) and general atrophy (60%). Focal lesions were most common in the frontal (42%) and temporal lobes (27%). About 20% of PDTBI patients demonstrated enlarged ventricles. By contrast, atrophy/volume loss is the most common type of MRI/CT finding for schizophrenia patients,

70% of the PDTBI patients in our study demonstrated EEG abnormalities.

INDIAN STUDIES

Sabhesan et al have emphasized the importance of pretraumatic personality, alcohol abuse, in the generic of delusions post head injury.

Subhasean et al have found that memory was found to be related to induces of severity of injury, particularly PTA presence of fracture of skull or early neurological deficits was not associated with poor performance.

Sabhasean et al has reported difference in memory scale scores in patients who continue to abuse alcohol after head injury and in patients who abstain from alcohol post head injury.

Sabhasean et al has reported 3 case studies in which patients have had delusions after head injury, one patient accused the doctor of performing vasectomy on him, the other patient occurred their relatives of stealing money, no site of injury and delusion association was made out and pharmacotherapy in the role of delusions could not be studied.

AIM

a) To study the incidence of psychiatric sequel in patients with head injury.

b) To compare the incidence of psychiatric sequel in head injury patients with that in non-head injury po7pulations.

c) To compare amongst the patients with head injury, the patients with depression and patients with personality disorder across demographic variables, injury site and duration.

d) To find cohort of high-risk patients.

HYPOTHESIS

1) There is no significant statistical difference in depression between head injury patients and control group.

2) There is no significant statistical difference in personality disorder between traumatic injury patients and controls.

3) There is no significant statistical difference in psychosis between traumatic brain injury patients and control.

4) There is no significant statistical difference in anxiety between head injury patients and control.

MATERIALS AND METHODS

Site of study

The study was carried out in neurosurgery out patient department of Stanley hospital. Patients were selected from follow up clinic of neurosurgery department conducted on Tuesdays, Thursdays and Saturday.

Selection of sample

50 patients with history of head injury and fulfilling the inclusion criteria were taken 50 attenders of other patients attending opd was taken, they were matched for age, sex and socio economic background.

Inclusion criteria

1. Age > 18 years
2. History of head injury
3. Moderate or severe.
4. Patient having received neurosurgical intervention.
5. 1 month after head injury

Exclusion Criteria.

1) Age < 18 years
2) Patients having delirium.
3) Patients having cognitive or physical deficits making interview impossible.

4) Patients having mild head injury

5) Patients having past history of psychiatric illness.

6) Patients having grossly psychotic status.

7) Patients having other medical or surgical problems.

8) Less than one month after head injury.

9) MMSE score below 23.

Tools Used

1) Proforma

2) DSM IV Criteria for personality disorder

3) MMSE– Mini Mental State Examination

4) HARS – Hamilton Anxiety Rating scale

5) HDRS – Hamilton Depression rating Scale.

HAMILTON DEPRESSION RATING SCALE (MATERIALS AND METHOD)

This scale is designed to measure the severity of illness of patients already suffering from depressive illness. It is obviously not a diagnostic instrument at is an observer rating scale hence raters must have sufficient clinical experience and judgment to be able to interpret the patients statements and reticence's about some symptoms and to compare them with other patient. The rater should use all the source of information (e.g. from relatives and nurses).

Hamilton Depression scale (HAMD) is a 21-item scale formatted for use with the general scoring sheet, the scale points vary from 3 to 5. The Hamilton Depression scale is one of the most widely used instruments for the clinical assessment of depressive states. The scale provides a simple way of assessing the severity of patient condition quantitatively and for showing changes in that condition. The symptoms are rated finely or coarsely, the former are on a five-point scale (0-4) where the numbers are equivalent to absent, doubtful or mild and obvious distinct or severe special problems regarding the female population are also considered separately.

HAMILTON RATING SCALE FOR ANXIETY

HAM – A developed by M. Hamilton, most widely utilized assessment scale for anxiety symptoms, and was originally intended to be used evaluate individuals who are already diagnosed with anxiety disorders.

The HAM – A consists of 14 items and like HAM – D, is heavily focused on somatic symptoms, each item is rated on a 0 – 4 scale.

ICD 10

F06 Other mental disorders due to brain damage and dysfunction and to physical disease

This category includes miscellaneous conditions causally related to brain dysfunction due to primary cerebral disease, to systemic disease affecting the brain secondarily, to endocrine disorders such as Cushing's syndrome or other somatic illnesses, and to some exogenous toxic substances (but excluding alcohol and drugs classified under F10-F19) or hormones; These conditions have in common clinical features that do not by themselves allow a presumptive diagnosis of an organic mental disorder, such as dementia or delirium. Rather, the clinical manifestations resemble, or are identical with, those of disorders not regarded as "organic" in the specific sense restricted to this block of the classification. Their inclusion here is based on the hypothesis that they are directly caused by cerebral disease or dysfunction rather than resulting 'from either a fortuitous association with such disease or dysfunction, or a psychological reaction to its symptoms, such as schizophrenia-like disorders associated with long-standing epilepsy.

The decision to classify a clinical syndrome here is supported by the following:

(a) evidence of cerebral disease, damage or dysfunction, or of systemic physical disease, known to be associated with one of the listed syndromes;

(b) a temporal relationship (weeks or a few months) between the development of the underlying disease and the onset of the mental syndrome;

(c) recovery from the mental disorder following removal or improvement of the underlying presumed cause;

(d) absence of evidence to suggest an alternative cause of the mental syndrome (such as a strong family history or precipitating stress).

Conditions (a) and (b) justify a provisional diagnosis; if all four are present, the certainty of diagnostic classification is significantly increased.

The following are among the conditions known to increase the relative risk for the syndromes classified here: epilepsy; limbic encephalitis; Huntington's disease; head trauma; brain neoplasms; extracranial neoplasms 'with remote CNS effects (especially carcinoma of the pancreas); vascular cerebral disease, .lesions, or malformations; lupus erythematosus and other collagen diseases; endocrine disease (especially hypo- and hyperthyroidism, Cushing's disease); metabolic disorders (e.g. hypoglycaemia, porphyria, hypoxia); tropical infectious

and parasitic diseases (e.g. trypanosomiasis); toxic effects of nonpsychotropic drugs (propranolol, levodopa, methyldopa, steroids, antihypertensives, antimalarials).

Excludes: mental disorders associated with delirium (F05.-) mental disorders associated with dementia as classified in FOO-F03

F06.0 Organic hallucinosis

A disorder of persistent or recurrent hallucinations, usually visual or auditory, that occur in clear consciousness and. may or may not be recognized by the subject as such. Delusional elaboration of the hallucinations may occur, but insight is not infrequently preserved.

Diagnostic guidelines

In addition to the general criteria in the introduction to F06 above, there should be evidence of persistent or recurrent hallucinations in any modality; no clouding of consciousness; no significant intellectual decline; no predominant disturbance of mood; and no predominance of delusions.

Includes : Dermatozoenwahn

Organic hallucinatory state (nonalcoholic)

Excludes: alcoholic hallucinosis (F10.52)

F06.1 Organic catatonic disorder

A disorder of diminished (stupor) or increased (excitement) psychomotor activity associated with catatonic symptoms. The extremes of psychomotor disturbance may alternate. It is not known whether the full range of catatonic' disturbances described in schizophrenia occurs in such organic states, nor has it been conclusively determined whether an organic catatonic state may occur in clear consciousness or whether it is always a manifestation of delirium, with subsequent partial or total amnesia. This calls for caution in making this diagnosis and for a careful delimitation of the condition from delirium. Encephalitis and carbon monoxide poisoning are presumed to be associated with this syndrome more often than other organic causes.

Diagnostic guidelines

The general criteria, for assuming organic etiology, laid down in the introduction to F06, must be met. In addition, there should be one of the following:

(a) stupor (diminution or complete absence of spontaneous movement with partial or complete mutism, negativism, and rigid posturing);

(b) excitement (gross hypermotility with-or without a tendency to assaultiveness);

(c) both (shifting rapidly and unpredictably from hypo- to hyperactivity). Other catatonic phenomena that increase confidence in the diagnosis are: stereotypies, waxy flexibility, and impulsive acts.

Excludes: catatonic schizophrenia (20.2) dissociative stupor (F44.2) stupor NOS (R40.1)

F06.2 Organic delusional [schizophrenia-like] disorder

A disorder in which persistent or recurrent delusions dominate the clinical picture. The delusions may be accompanied by hallucinations but are not confined to their content. Features suggestive of schizophrenia, such as bizarre delusions,.hallucinations, or thought disorder, may also be present.

Diagnostic guidelines

The general criteria for assuming an organic etiology, laid down in the introduction to F06, must be met. In addition, there should be delusions'(persecutory, of bodily change, jealousy, disease, or death of the subject or another person). Hallucinations, thought disorder, "or isolated catatonic phenomena may be present. Consciousness and memory must not be affected. This diagnosis should not be made(if the presumed evidence of organic causation is nonspecific or limited to findings such as enlarged cerebral ventricles (visualized on computerized axial tomography) or "soft" neurological signs.

Includes: paranoid and paranoid-hallucinatory organic states schizophrenia-like psychosis in epilepsy

Excludes: acute and transient psychotic disorders (F23. —)

drug-induced psychotic disorders (Flx.5)

persistent delusional disorder (F22. -)

schizophrenia (F20.-)

F06.3 Organic mood [affective] disorders

Disorders characterized by a change in mood or affect, usually companied by a change in the overall level of activity. The only criterion for inclusion of these disorders in this block is their presumed direct causation by a cerebral or other physical disorder whose presence must either be demonstrated independently (e.g. by means appropriate physical and laboratory investigations) or assumed i the basis of adequate history information. The affective disorder must follow the presumed organic factor and be judged not to present an emotional response to the patient's knowledge of having, or having the symptoms of, a concurrent brain disorder.

postinfective depression (e.g. following influenza) is a common :ample and should be coded here. Persistent mild euphoria not amounting to hypomania (which is sometimes seen, for instance,

association with steroid therapy or antidepressants) should not : coded here but under F06.8.

Diagnostic guidelines

In addition to the general criteria for assuming organic etiology, id down in the introduction to F06, the condition must meet the requirements for a diagnosis of one of the disorders listed under F30-F33.

Excludes: mood [affective] disorders, nonorganic or unspecified (F30-F39) right hemispheric affective disorder (F07.8)

The following five – character codes might be used to specify the clinical disorder :

F06.30 Organic manic disorder

F06.31 Organic bipolar affective disorder

F06.32 Organic depressive disorder

F06.33 Organic mixed affective disorder

F06.32 Organic depressive disorder F06.33 Organic mixed affective disorder

F06.4 Organic anxiety disorder

A disorder characterized by the essential descriptive features of a generalized anxiety disorder (F41.1), a panic disorder (F41.0), or a combination of both, but arising as a consequence of an organic disorder

capable of causing cerebral dysfunction (e.g. temporal lobe epilepsy, thyrotoxicosis, or phaechromocytoma).

Excludes: anxiety disorders, nonorganic or unspecified (F41.-)

F06.5 Organic dissociative disorder

A disorder that meets the requirements for one of the disorders in F44. — (dissociative [conversion] disorder) and for which the general criteria for organic etiology are also fulfilled (as described in the introduction to this block).

Excludes: dissociative [conversion] disorders, nonorganic or unspecified (F44.-)

F06.6 Organic emotionally labile [asthenic] disorder

A disorder characterized by marked and persistent emotional incontinence or lability, fatiguability, or a variety of unpleasant physical sensations (e.g. dizziness) and pains regarded as being due to the presence of an organic disorder. This disorder is thought to occur in association with cerebrovascular disease or hypertension more often than with other causes.

Excludes: somatoform disorders, nonorganic or unspecified (F45.-)

F06.7 Mild cognitive disorder

This disorder may precede, accompany, or follow a wide variety of infections and physical disorders, both cerebral and systemic (including HIV infection). Direct neurological evidence of cerebral involvement is not necessarily present, but there may nevertheless be distress and interference with usual activities. The boundaries of this category are still to be firmly established. When associated with a physical disorder from which the patient recovers, mild cognitive disorder does not last for more than a few additional weeks. This diagnosis should not be made if the condition is clearly attributable to a mental or behavioural disorder classified in any of the remaining blocks in this book.

Diagnostic guidelines

The main feature is a decline in cognitive performance. This may include memory impairment, learning or concentration difficulties. Objective tests usually indicate abnormality. The symptoms are such that a diagnosis of dementia (FOO — F03), organic amnesic syndrome (F04) or delirium (F05. —) cannot be made.

Differential diagnosis. The disorder can be differentiated from postencephalitic syndrome (F07.1) and postconcussional syndrome

(F07.2) by its different etiology, more restricted range of generally milder symptoms, and usually shorter duration.

F06.8 Other specified mental disorders due to brain damage and dysfunction and to physical disease

Examples are abnormal mood states occurring during treatment with steroids or antidepressants.

Includes: epileptic psychosis NOS

F06.9 Unspecified mental disorder due to brain damage and dysfunction and to physical disease.

MMSE - Mini Mental State Examination

The MMSE is a 30 point cognitive test developed in the mid 1970s to provide a bedside assessment of a broad array of cognitive functions including orientation, attention, memory, construction and language. For patients with extensive education who may score 30 out of 30 despite clear evidence of functional decline, a more difficult cognitive tests full neuropsychological battery may be done.

RESULTS AND OBSERVATION

SOCIO DEMOGRAPHIC CHARACTERISTICS OF THE SAMPLE

Age of the persons in this study group was about 18 years, males constituted the majority of patients in this study. As age and sex matched controls were taken for study their percentage was same as for cases.

Among cases majority were married (82%). Among control subjects who were matched for age and sex (90%) were married.

Among cases majority (74%) were from nuclear families (26%) were from joint families. Among controls (58%) were from nuclear families and 42% from joint families.

TABLE 1
AGE DISTRIBUTION

Age Group (yrs)	No. of Cases	Controls	Chi-square	P value
20-30	11 (22.0%)	12 (24.0%)	1.58	0.67 not significant
30-40	22 (44.0%)	26 (52.0)		
40-50	11 (22.0%)	9 (18.0%)		
> 50	6 (12.0%)	3 (6.0%)		
Total	50	50		

There was no statistical significance difference between cases and control in age distribution.

TABLE – 2

SEX DISTRIBUTION

Type of Subjects	Male	Female	Total	Chisquare	P value
Patient	46 (92.0%	4 (8.0%)	50	0.71	0.40 NS
Control	48 (96.0%)	2 (4.0%)	50		
Total	94 (94.0%)	6 (6.0%)	100		

There was no statistical significance difference between cases and control in sex distribution. Males were predominant (94%).

TABLE – 3

RELIGION

Religion	Hindu	Muslim	Christian	Total	X^2	P value
Patient	46 (92%)	3 (6%)	1 (2%)	50	1.51	0.47
Control	45 (90%)	5 (10%)	-	50		

TABLE – 4
MARITAL STATUS

Marital Status	Patients	Control	X^2	P value
Married	41(82%)	45 (90%)	2.0	0.57
Unmarried	7 (14%)	4 (8%)		
Separated	1 (2%)	0		
Divorced	1 (2%)	1(2%)		

There was no significant statistical difference between patients and controls.

TABLE – 5
FAMILY SYSTEM

Family	Patients	Controls	X^2	P value
Joint	13 (26%)	21 (42%)	2.85	0.009
Nuclear	37 (74%)	29 (58%)		
Total	34 (34%)	66 (66%)		

TABLE – 6

Education	Patients	Controls	X^2	P value
Illiterate	8(16%)	7 (14%)	1.75	0.78 Not significant
Primary	5 (10%)	2 (4%)		
Middle	18 (36%)	22 (44%)		
H.S.C.	16 (32%)	16 (32%)		
College	3 (6%)	3 (6%)		

36.0% of study group patients had a middle school educational qualification, and controls (44.0%) were similarly matched, no statistically significant difference.

TABLE – 7
EMPLOYMENT

Occupation	Patients	Controls	X^2	P Value
Employed	33 (66.0%)	40 (80.0%)	2.49	0.11
Unemployed	17 (34.0%)	10(20.0%)		Not significant

Study group (66.0%) were employed and similarly matched with control group.

TABLE – 8
INCOME

Income (Rs.)	Patients	Controls	X^2	P Value
0-500	23 (46.%)	13 (26%)	4.52	P = 0.21
500-1000	17 (34%)	25 (50%)		Not significant
1000-2000	2 (4%)	10 (20%)		
> 2000	8 (16%)	2 (4%)		

No statistically significant difference between cases and controls. Majority of patients and controls belonged to 0 -1000 income group.

TABLE – 9

Housing	Patients	Controls	X^2	P Value
Own	16(32%)	17 (34%	0.04	P = 0.82
Rental	34 (68%)	33(66%)		No significant

Majority of patients were coming from housing mostly rental in nature.

TABLE - 10
TYPES OF INJURY

Types of Injury Cause	No. of Cases	Percentage
RTA	33	66.0%
Assault	4	8.0%
TTA	2	4.0%
Fall	11	22.0%

RTA (Road Traffic Accident) constituted the majority among causes of head injury, with fall from heights, causing 22% of the injury.

TABLE – 11
SEVERITY

Severity	No. of Cases	Percentage
Moderate	45	90.0%
Severe	5	10.0%

TABLE – 12

Site	No. of Cases	Percentage
Frontal	17	34.0%
Parietal	6	12.0%
Temporal	9	18.0%
Occipital	4	8.0%
Parietoccipital	1	2.0%
Frontoparietal temporal	2	4.0%
Frontoparietal	3	6.0%
Frontotemporal	8	16.0%

In our study (34%) had injuries sustained in frontal region.

TABLE – 13
TIME AFTER INJURY

Time after injury	No. of Cases	Percentage
1-6 months	17	34.0%
6-12 months	19	38.0%
1-3 yrs	12	24.0%
> 3 yrs	2	4.0%

Table - 14
PSYCHIATRIC SEQUALE POST TBI

Psychiatric sequale	Group				Significance
	Control(50)		Study(50)		
	n	%	n	%	
Absent	44	88.0%	19	38.0%	χ^2=26.81 P=0.001 (S)
Anxiety	1	2.0%	2	4.0%	χ^2=0.34 P=0.55 (NS)
Depression	5	10.0%	12	24.0%	χ^2=3.84 P=0.05 (S)
Psychosis	-	-	2	4.0%	χ^2=0.51 P=0.48 (NS)
Personality disorder	-	-	4	8.0%	χ^2=4.17 P=0.04 (S)
Anxiety with personlitydisorder	-	-	3	6.0%	χ^2=3.09 P=0.08 (NS)
Depression with personality disorder	-	-	5	10.0%	χ^2=5.26 P=0.02 (S)
Depression with anxiety	-	-	3	6.0%	χ^2=3.09 P=0.08 (NS)

Among cases 62% qualified for psychiatric diagnosis and among controls 12% qualified for psychiatric diagnosis. Among the psychiatric diagnosis of cases majority consisted of depression (24.0%) 12 persons, 5 persons (10.0%) had mixed diagnosis of both depression and personality disorder and 4 persons (8%) had personality disorder, 3

persons had (6%) anxiety with personality disorder, 2 persons had anxiety (4%) and 2 persons had psychosis (4%).

Among the head injury patients, depressed patients were 12 in number, they were analysed over sociodemographic variables.

TABLE - 15
DEPRESSION AFTER TBI

a) Age Distribution

Age	Depression				Significance
	Not depressed		Depressed		
	n	%	n	%	
20-30	8	21.1%	3	25.0%	
30-40	17	44.7%	5	41.7%	$\chi 2$=0.33
40-50	8	21.1%	3	25.0%	P=0.96 (NS)
>50	5	13.2%	17	8.3%	
Total	38	100.0%	12	100.0%	

b) Sex Distribution

Sex	Depression				Significance
	Not depressed		Depressed		
	n	%	n	%	
Male	36	94.7%	10	83.3%	$\chi 2_{vates}$=0.43
Female	2	5.3%	2	16.7%	P=0.50 (NS)
Total	38	100.0%	12	100.0%	

c) Occupation

Occupation	Depression				Significance
	Not depressed		Depressed		
	n	%	n	%	
Employed	24	63.0%	9	75.0%	$\chi 2_{vates}$=0.16
Unemployed	14	37.0%	3	25.0%	P=0.68 (NS)
Total	38	100.0%	12	100.0%	

e) Severity of Injury

Severity of injury	Depression				Significance
	Not depressed		Depressed		
	n	%	n	%	
Moderate	34	89.4%	4	33.3%	$\chi 2=15.7$
Severe	4	10.6%	8	66.7%	P=0.001 (S) OR
Total	38	100.0%	12	100%	(95%CI)= 17(3-70)

Among the depressed patients, majority belonged to severe head injury. This is in accordance with Jorge et al, but other studies have found no association between head injury severity and depression.

f) Time after injury

Time	Depression				Significance
	Not depressed		Depressed		
	n	%	n	%	
1-6 months	10	26.3%	8	75.0%	$\chi 2=8.32$ P=0.04 (S)
6-12 months	15	39.5%	4	25.0%	
1-3 yrs	11	28.9%	0	-	
> 3 yrs	2	5.3%	0	-	
Total	38	100.0%	12	100.0%	

There is a significant statistical difference between head injury patients regarding duration of head injury and appearance of depression.

f) Site of Injury

Site	Depression			
	Not depressed		Depressed	
	n	%	n	%
Frontal	12	31.6%	5	41.7%
Parietal	4	10.5%	2	16.7%
Temporal	6	15.8%	3	25.0%
Occipital	3	7.9%	1	8.3%
Parietoccipital	1	2.6%		
Frontoparietal temporal	1	2.6%	1	8.3%
Frontoparietal	3	7.9%		
Frontotemporal	8	21.1%		
Total	38	100.0%	12	100.0%

TABLE - 16
PERSONALITY DISORDER

a) Age Distribution

Age	Personality				Significance
	No disorder		Disorder		
	n	%	n	%	
20-30	9	19.6%	2	50.0%	χ^2=4.09 P=0.25 (NS)
30-40	22	47.8%			
40-50	10	21.7%	1	25.0%	
>50	5	10.9%	1	25.0%	
Total	46	100.0%	4	100.0%	

b) Sex

Sex	Personality				Significance
	No disorder		Disorder		
	n	%	n	%	
Male	42	91.3%	4	100.0%	χ^2=0.38 P=0.54 (NS)
female	4	8.7%			
Total	46	100.0%	4	100.0%	

c) Time after injury

Time	Personality				Significance
	No disorder		Disorder		
	n	%	n	%	
1-6 months	16	34.7%	2	50.0%	$\chi 2=1.71$ P=0.63 (NS)
6-12 months	17	37.0%	2	50.0%	
1-3 yrs	11	23.9%			
> 3 yrs	2	4.3%			
Total	46	100.0%	4	100.0%	

There is no significant statistical difference in our study. Fedroff et al found no correlation between time since injury and appearance of personality disorder, but other studies have shown correlation between appearance of syndrome and time since injury.

d) Site

Site	Personality			
	No disorder		Disorder	
	n	%	n	%
Frontal	14	30.4%	3	75.0%
Parietal	6	13.0%		
Temporal	9	19.6%		
Occipital	4	8.7%		
Parietoccipital	1	2.2%		
Frontoparietal temporal	2	4.3%		
Frontoparietal	2	4.3%	1	25.0%
Frontotemporal	8	17.4%		
Total	46	100.0%	4	100.0%

DISCUSSION

a) The two groups of patients and controls were compared over various socio demographic standards for comparison. They were compared over variables of age, sex, marital status, religion, employment status, per capita income educational status, family structure and housing type. The two groups were compared statistically and no significant statistical difference was found between the two groups. So the two groups were equally matched for comparison.

b) Psychiatric diagnosis were made in cases and controls by using ICD 10 diagnostic criteria and personality disorder was made using DSM – IV criteria, chi square test was used with one degree of freedom and Yates correction wherever necessary to statically compare the sequel of psychiatric disorders between cases and controls 'P' value obtained was <0.05 indicating that psychiatric sequel incidence is statistically significant in patients with Traumatic brain injury compared to control population.

In this study incidence of psychiatric sequel in head injury patients was 62% (range 48% - 75%) this is in accordance with study by **Koponen et al** who reported incidence of 48.3%, and

similar to findings by **Rao et al** who reported a variety of psychiatric disturbances ranging from subtle deficits in cognition mood and behavior to severe disturbance that cause impairment in social, occupational and interpersonal functioning

This study shares similarities with studies by **Soomitra deb et al** which reported cases in which dual diagnosis was made in this study 3 patients had Anxiety with personality disorder; 5 patients had depression and organic personality disorder, and 3 patients had depression with anxiety and significant stastical difference had been reported in this population the personality disorder diagnosed in 5 patients were 3 had apathetic and 2 had aggressive behavior, this is in accordance with study done by **tatano et al** who found that 33.7% of TBI patients demonstrated significant aggression more frequent in depressive patients and also had poor social functioning which was not assessed in this study.

In this study depression was present in 12 out of 50 patients and tested above 14 on HDRS compared with 5 of study population, p value obtained was 0.05 (significant), and depression was compared with not depressed among sociodemographic variables.

Shoumitradeb et al had reported incidence of 12.8%. The first report of a higher rate of ICD-10 psychiatric diagnosis among adult patients suffering from depression post TBI was made by this author

Study by Rapoport et al had reported 28.4% of patients with depression and he had also reported a high association with injures to anterior temporal and frontal lobes, which is similar in this study.

One study by Reekum et al had reported female predominance in post TBI depression; incidence has been studied to be 44%. Jorge et al had described alcohol abuse and mood disorder to be a co-occurring condition, and he had reported comorbid depression with anxiety (76.7%) and aggressive behavior (56.7%). O'Caroll RE in his study found no correlation between severity of head injury and anxiety depression or psychosexual changes following head injury. On comparing amongst the head injury patients those with depression against those without depression over various variables, it was seen that

i. Age, when age group was broken up, 5 patients were depressed in age group of 30-40 years. The head injury may have acted as a stressor to bring about the depressive symptoms in patients who were predisposed the same.

ii. The coping resources decelerate as age progresses.

iii. The burden of responsibilities is much higher on the elderly population, but in this study it was not replicated.

iv. No statistically significant differences were observed across the sociodemographic variables.

v. There was a statistically significant difference in the number of patients found depressed, in first six months after head injury, as compared to non-depressed subjects, findings are consistent with Jorge et al who had reported 42% of TBI patients had suffered from depression and majority had been diagnosed in the first 3 months after injury.

PERSONALITY DISORDER

In this study, personality disorder was diagnosed in 4 patients using the DSM IV TR criteria, compared with the control group it was statistically significant.

Patients were compared across sociodemographic variables and no significance was noted and of 4 patients 3 had aggressive personality, in accordance with study by Koponen et al who reported a rate of 5.9% to 13.5% in our study one person above 50 had personality disorder considering that personality disorder declines with age (Cohen et al, Pogel BS et al), the most common disorder reported by Hibbard et al was avoidant and paranoid (26%) personality disorder, it seems that

traumatic brain injury can expose some individuals to social anxiety, suspiciousness and detachment and borderline personality disorder is to be the most prevalent disorder after traumatic brain injury in a study by Hibbard et al only organic personality disorder was diagnosed in our study.

Koponen et al reported a incidence of 15.0% for organic personality disorder and severity of brain injury was not associated with the presence of personality syndrome, also reported by Franulic et al.

2 patients had anxiety (4%), since the sample population is small, patients were not assessed across all variables, but other studies have found anxiety after head injury high by Ocarrole RE (26%). Other anxiety disorders panic disorder was diagnosed in 8.3% of the patients, since social support is high, patients might have had lesser incidence, in the acute stages due to fear about consequences, and circumstances of injury, patients reported anxiety, PTSD was increased according to Wright JC et al, but Sbordone et al found PTSD absent in cases of mild head injury.

In this study 5 patients of 50 had coexisting depression with personality disorder; P value was 0.02 (significant) (10.0%).

The other psychiatric sequale like anxiety with personality disorder (6%), depression with anxiety (6%) (Not significant).

HYPOTHESIS 1

There is no significant statistical difference in depression between head injury patients and control group.

	Control		**Study**		
	N	**%**	**N**	**%**	
Depression	5	10.%	12	24%	$X^2 - 3.84$ P = 0.05

In our study depression was 24.0% (n =12) amoung patients with Traumatic Brain Injury.

The first report of a higher rate of depressive episode among patients with Traumatic Brain Injury compared with controls was by Shoumitra Dep et al who reported a rate of 13.9% compared with 2.1% of general population and another study by Salla Koponen et al reported that the most common Axis I disorder after traumatic brain injury were major depression (26.7%), also a study by Jarge et al reported a incidence of 33% (30 of 91 patients) during the first year of sustaining a traumatic brain injury.

HYPOTHESIS 2

There is no significant statistical difference between traumatic Injury patients and controls.

	Control		Study		
	N	**%**	**N**	**%**	
Personality Disorder	-	-	4	(8%)	$X^2 = 4.17$ P = 0.04 Significant

In this study 8.0% (4 patients of 50) showed personality changes and 3 patients showed aggressive changes and 1 patient showed apathetic changes.

This is in accordance with studies by Graffman et al and Federoff et al.

Persinger et al in his study on personality changes following head injury found that injures to temporofrontal regions was more commonly associated with personality changes.

HYPOTHESIS 3

There is no significant statistical difference in psychosis between traumatic brain injury patients and control.

	Control		Study		
	N	**%**	**N**	**%**	
Psychosis	-	-	2	4%	$X^{2} = 0.51$ P = 0.48 Not significant

In this study even though percentage of psychosis in study population was high, no significant difference was made, this is in contrast to other studies by Salla Koponen et al which reported a rate of 8.3% but ECA survey had reported 0.7%, another study by Achte et al had a rate of 9% even though in this study prevalence is high no significant difference could be made might be due to the smaller size of sample and the other studies were done on western population, but similarities were noted like the study done by Daryl Fujii et al who reported age of onset of psychosis after head trauma was $33.4^{\pm}$ 15.4 years in this study age of onset was around 30-40 years and the symptom reported in study by Fujii et al was persecutory in this study it was paranoid in both patients, and onset of psychosis after traumatic brain injury was within 1 year similar to this study. Study by Koponen et al had reported paranoid features.

HYPOTHESIS 4

There is no significant statistical difference in anxiety between head injury patients and control.

	Control		Study		
	N	**%**	**N**	**%**	
Anxiety	1	5%	2	4%	$X^2 = 0.34$ P = 0.55 Not significant.

The difference in proportions between the two groups is statistically insignificant. 2 patients were found to have scores > 14 on HARS.

This is in contrast to various studies showing incidence of anxiety after head injury is higher than normal population, in a study done by Robert Van Reekum et al has reported a incidence of 9.1% of generalized anxiety disorder, in which evidence of temporal sequence was consistently positive, in the same study 6.4% had obsessive compulsive disorder, 9.2% for panic disorder, 14.1% for post traumatic stress disorder.schoenhuber and gentilini(24) compared rate of GAD,no significant difference was detected

SUMMARY

The present study is an attempt to find out the incidence of psychiatric sequel and the significance of the same in head injury patients.

The sample in this study consisted of fifty cases from outpatient department of Stanley's Neurosurgery department and fifty control from relatives and friends of other patients attending opd for other conditions, matched individually with cases for age and sex, after obtaining informed consent.

There was no significant difference between cases and control, with regard to sociodemographic variables.

The psychiatric sequel in head injury patients is significantly higher than the general population.

The incidence of depression, personality disorder, and depression and personality disorder combined are higher after head injures.

Amongst the head injury patients those having significant depression were of,

- Age group of 30-40 years
- Had moderate head injury
- Was found more during the first 6 months of injury

So patient population of head injures about 30 years, moderate head injury, and greater amount of physical damage and during first six months of injury should be given a special attention and assessed for development of depression. Almost 40-50% of patients with moderate or severe head injury develops clinical depression. Almost 14% of deaths post head injury are due to suicide.

Looking at the statistics of present study and correlating it with present literature, this particular cohort of patients should be given special attention.

Similarly dual diagnosis of Anxiety with personality disorder, depression with personality disorder, depression with anxiety was present but looking at the sample size, it would be unwise to make any statements regarding the same except that depression and aggressive behavior has been found in quite a few studies and significant statistical difference was noted.

Regarding psychosis even though sample reported 4.0%, it was statistically insignificant but higher than in general population but sample, size, the number of patients it would be unwise to make any statements regarding the same.

4 patients showed personality changes as compared to non in the control group. When assessed on DSM IV organic personality scale. Site

of injury was frontal in 3 of them, care should be taken to assess patient's personality change and referral to psychiatry setup immediately.

CONCLUSION

- Psychiatric sequelae is more in head injury patients when compared with non head injury population.
- Depression is significantly more in head injury population.
- Personality disorder is more frequent in head injury population.
- Moderate to severe head injury has significant correlation with psychiatric sequelae.
- Psychiatric sequelae is more in the initial few months after injury.
- Injury to frontal region has significant association with personality disorder.
- Younger age group are more at risk for psychiatric sequelae.

LIMITATIONS OF THE STUDY & SUGGESTIONS

1. In the present study where aim was a comparatively broader one, the wealth of detailed information on specific areas could not be a assessed.
2. Small sample size.
3. Cross sectional nature of sample.
4. Lack of systematic neuroradiological investigations at the time of injury information on the nature and location of brain injury and consequently on their association with development of psychiatric disorders remained insufficient.

In this study alcohol dependence / abuse was not taken in to study, but literature reveals association between alcohol dependence and depression which needs to be explored.

Neuroimaging studies of patients with psychiatric sequel post traumatic brain injury might reveal a lot of information about the etiology.

Results suggest that traumatic brain injury can cause decades of or even permanent vulnerability to psychiatric disorders in some individuals.

Personality disturbances which were common among our patients, can be difficult to detect and may impair compliance with rehabilitation. Therefore psychiatric evaluation and followup should be included in the routine treatment of head injury.

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PROFORMA

1. Name
2. Age (Year)
3. Sex (Male/Female)
4. Religion (Hindu/Muslim/Others)
5. Marital Status (Married/Un Married/Others)
6. Family Type (Joint / Nuclear)
7. Educational Status (Uneducated/Educated [Details])
8. Occupation (Employed / Unemployed [Details])
9. Per Capital Income (Kuppuswamy Scale Rs. Per Month)
10. Housing (Own/Rent)
11. Injury Details
    - Cause
    - Severity (Initial score on (Cs on recod)
    - Site of injury
    - Neurosurgical interventions
12. Detailed present/ past/ personal / family/ personality history
13. Physical examination
14. Mental status examination
15. MMSE
16. HARS
17. HDRRS
18. DDSM IV Criteria

APPENDIX 1

HAMILTION DEPRESSION RATING SCALE

1) Depression (0-4) (gloomy attitude, pessimism about future, feelings of hopelessness, tendency to weep)

0. not depressed 1. doubtful, trivial 2. mild (occasional weeping)

3. moderate (frequent weeping) 4. Severely depressed

2) Guilt (0-4) (pathological guilt)

0. absent 1. feelings of self reproach 2. ideas of guilt

3.illness might be punishment 4.delusion of guilt

3) suicide (0-4)

0.absent 1. life not worth living 2. wishing he were dead

3. suicidal ideas, half hearted attempts 4. Serious suicidal attempts

4) initial insomnia (0-2) (difficulty in getting to sleep)

0.not present 1. nil, trivial, infrequent 2. obvious and severe symptoms

5) middle insomnia (0-2) (disturbed sleep during the night)

0.not present 1. nil, trivial, infrequent

6) delayed insomnia (0-2) (early morning awakening)

0.not present 1. nil, trivial, infrequent 2. obvious and severe symptoms

7) Work and interest (0-4) (loss of interest in and decreased performance at work or in home duties) do not rate fatigue or energy her

0.not disturbance 1. doubtful, trivial 2. mild 3. moderate

4. Severe- unable to carry on

8) Retardation (0-4)

0. absent	1. slight flattening of affect, fixity of expression	2. monotonous voice, delay in answering questions, tendency to sit motionless,
3. retardation prolongs interview to an extreme degree.		4. to a degree which makes interview impossible

9) Agitation (0-4) (this may co-exist with retardation)

0. not present	1. Fidgetiness at interview	2. obviously restless, picking at hands and clothes
3. had to get up during interview		4. cannot stay still, tearing clothes

10) Anxiety (psychic)(0.4) (tension, difficulty in relaxing, irritability, worrying over trivial matter, apprehension, feelings of panic, fears, difficulty in concentration etc. rate these symptoms of present illness and not as features or previous deposition).

0. absent	1. doubtful, trivial	2.mild
3. moderate		4.severe

11) Anxiety (somatic)(0.4) (effects of autonomic over activity, attacks of giddiness, blurring of vision and tinnitus).

0. absent	1. doubtful, trivial	2.mild
3. moderate		4.severe

12) Gastrointestinal symptoms (0-2) (loss of appetite, constipation, heavy feelings in abdomen, differentiate from symptoms which could be rated under anxiety above)

0. absent	1. nil, trivial, infrequent	2. obvious and severe symptoms

13) general somatic symptoms (0-2) (fatigability, loss of energy, diffuse and ill-defined muscle aches, heaviness of limbs)

0. absent	1. nil, trivial, infrequent	2.obvious and severe symptoms

14) loss of libido (0-2) (assess deterioration obviously related to present illness

0. absent | 1. mil, trivial, infrequent | 2. obvious and severe symptoms

15) hypochondriasis (0-4)

0. absent | 1. trivial,doublful, some preoccupation with bodily functions | 2. much preoccupation with physical symptoms and with thoughts of organic disease

3. strong convictions of presence of organic disease to account for symptoms | 4. delusions, hallucinations of rotting, blockage,etc.

16) loss of insight (0-2)

0. has full insight | 1. doubtful,mild , some denial | 2. lacks insight

17) loss of weight (0-2)

0. no change, or increase in weight | 1. doubtful, slight loss | 2. obvious, severe loss

18) diurnal variation (0-2)

0. not present | a) symptoms worse in morning | b) symptoms worse in evening

1. doubtful, present to a mild degree | 2.clear presence

19) derealisation and depersonalization (0-4) (differentiate from lack of concentration or interest)

0. not present patient does not understand feelings from the question asked	1. recognises feelings when asked but only experiences these mildly or doubtful	2. recognises feelings when asked and experiences them frequently.
3.asserts that these feelings are present as part of his illness.	4. claims that these feelings are an important symptom of his illness.	

20) paranoid symptoms (0-4) (check affirmative answers carefully. Differentiate from guilt feelings. Include attitude of suspicion and malevolence imputed to others)

0. not present.not elicited	1. doubtful, trivial suspicion	2. Thinks others may wish him harm
3. delusions that others may wish him harm	4.paranoid hallucinations	

21) obsessional symptoms (0-2) (differentiate from preoccupations with depressive thoughts, ideas of guilt, hypochondriasis, paranoid thinking. Patient recognizes thoughts as being alien to normal thoughts and feelings, as producing distress and always struggles against them)

0. no evidence	1. doubtful or to a mild degree	2. definitely present to a severe degree

22) helplessness'(0-4)

0. not present	1. subjective feeling which are elicited only by inquiry	2. patients volunteers his helpless feelings
3. requires urging , guidance and reassurance to accomplish word chores or personal hygiene	4. requires physical assistance for dress, grooming, eating beside tasks or personal hygiene	

23) Hopelessness (0-4)

0. not present	1. intermittently doubts that things will improve" but can be reassured	2. consistently feels "hopeless" but accepts reassurances.
3. expresses feelings of discouragement, despair, pessimism about future, which cannot be dispelled.	4. spontaneously and inappropriately perseverant, "I'II never get well or its equivalent.	

24) worthlessness (0-4) (ranges from mild loss of esteem, feelings of inferiority, self depreciation to delusional notions of worthlessness)

0. not present	1. indicates feelings of worthlessness only on questioning (loss of self-esteem),	2. spontaneously indicates feelings of worthlessness (loss of self esteem)
3. different from 2 by degree: patient volunteers that he is "no good", "inferior", etc.	4. delusional notions of worthlessness - i.e. " I am a heap of garbage" or its equivalent	

APPENDIX 2

Max Hamilton's anxiety rating scale

Symptoms of anxiety states

1. Anxious Mood
   Worries
   Anticipation of the worst apprehension (fearful anticipation)
   Irritability
2. Tension
   Feeling of tension
   fatiguability
   Inability to relax
   Startle response
   Moved to tears easily
   trembling
   Feelings of restlessness
3. Fears
   Of dark
   Strangers
   Being left alone
   Large animals, etc.,
   Traffic
   Crowds.
4. Insomnia :
   Difficulty in falling asleep,
   Broken sleep,
   ursatisfying sleep and fatigue on walking
   Dreams
   Night mares
   Night terrors
5. Intellectual (cognitive)
   Dificulty in concentration
   poor memory
6. Depressed mood
   Loss of interest
   Lack of pleasure in hobbies
   Depression early waking
   Diurnal swing
7. General somatic (muscular)
   Muscular pains and aches
   Muscular stiffness Muscular twitchings
   clonic jerks
   Grinding of teeth
   Unsteady voice
8. Cardiovascular symptoms
   Tachycardia
   Palpitations
   Pain in chest
   Throbbing of vessels
   Fainting of feelings missing beats
9. Respiratory Symptoms
   Pressure or constriction in chest
   Choking feeling
   Sighing
   dyspnoea

10. Gastro – intestinal symptoms
Difficulty in swallowing
Wind
Dyspepsia:
Pain before and after meals
Burning sensation fullness
Water brash
Nauseas
Vomiting
Sinking feelings
Working in abdomen borbory gmi
Looseness of bowels loss of weight
Constipation

11. Genito – urinary symptoms:-
Frequency of micturition
urgency of micturition
Amenorrhea
Menorrhagia
Development of frigidity
Ejaculation pracox
Loss of erection
Impotence

12. Autonomic symptoms: -
Dry mouth flushing
Pallor
Tendency to sweat
Giddiness
Tension headache
Raising of hair

13. Behavior at interview (general)
Tense, not relaxed
Fidgeting:- hands, picking fingers

14. Behaviour (Psysiological)
Swallowing belching
High resting pulse rate
Respiration rate over 20/min
Brisk tension jerks
Tremor
Dilated pupils
Exophthalmoses
Sweating
Eye-lid twitching

15
General Somatic (sensory)
Tinnitus
Blurring of vision Hot and Cold
Flushes
Feeling of weakness
Pricking sensations

16.

APPENDIX 3

1) Anxious mood
2) Tension
3) Fears
4) Insomnia
5) Intellect
6) Depressed mood
7) Somatic general (muscular & sensory)
8) Cardiovascular system
9) Respiratory system
10) Gastro – intestinal system
11) Genito – urinary system
12) Autonomic system
13) Behavior at interview

General comments:

0 Is none
1 is mild
2 is moderate
3 is severe
4 grossly disabling

0-5.1 no anxiety
6-14 Mild anxiety
15 major anxieties

APPENDIX 4
MINI MENTAL STATE EXAMINATION (MMSE)

Now I would like to ask you some questions to check your memory and concentration. Some of them may be easy and some of them may be difficult.

1. is it morning or afternoon or evening? 0/1

2. what day of the week is it today? 0/1

3. what date is it today?(Tamil or English calendar) 0/1

4.which month is this?(Tamil or English calendar) 0/1

5. what season of the year is it? 0/1

6.which post office does this village come under? 0/1

7.which district does this village fall under? 0/1

8. which village is this? 0/1

9. which block is this?(which numbered area is this) 0/1

10.wbich place is this?(whose house -is this) 0/1

11. I went to Madurai and brought back three things; 0/1

they are mango...., a chair...., and a Coin.

Can you tell me what are the three things I brought from madurai? 0/1

Remember the names of the three things I have brought from madurai As I am going to ask you to name them after some time.

12. now can you tell me the names of the days of week starting from Sunday now can you tell me the names of these days backwards? For example Saturday comes before Sunday. And before that,, and before that, and before that,,, and before that,, and before that,,, and before that. l/2/3/4/5

13. What are the names of the three things, which I told you I have brought from madurai? 0/1

14. 0/1

15. . 0/1

16. (show wrist watch) what is this? 0/1

17. (show pen) what is this? 0/1

18. Now 1 am going to say something, listen carefully and repeat it exactly as I say it, after I have finished.

"neither this, nor that." 0/1

19. now I am going to ask you a different type of question. Now look- at my face and do exactly what I do.

Interviewer:* close your eyes for 2 seconds and then open them.

Completed: no/yes 0/1

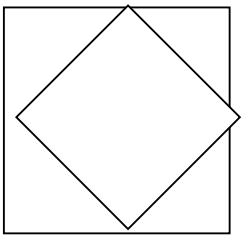
20. Now I am going to give you a piece of paper with which you have to do exactly what I ask you to do. first take the paper in your right hand; then with both hands fold into half once and then give ihe paper back to me.

Right hand	no/yes	0/1
Folds	no/yes	0/1
Returns	no/yes	0/1

21. Now say a line about your house.

Cue given	no/yes

22. Here is a drawing. You must copy this drawing exactly as shown in the space provided here.



To be used as an alternative test of attention (instead of days backward), if desired.

23. Now try to do some calculations.

If a man has Rs. 20 for bus fare and every day he spends Rs. 3 on his bus fare, after spending the first days bus far, he will be Rs. 17. How much money will be left after next day bus fare? After next days bus fare?

After next days bus fare? After next days bus fare? 0/1/3/4/5

Maximum score = 30

APPENDIX 5

PERSONALITY CHANGE DUE TO

[Indicate the General Medical Condition]

a) A persistent personality disturbance that represents a change from the individual's previous characteristic personality pattern. (In children, the disturbance involves a marked deviation from normal development or a significant change in the child's usual behaviour patterns lasting at least 1 year).

b) There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct physiological consequence of a general medical condition.

c) The disturbance is not better accounted for by another mental disorder (including other Mental Disorders due to a General Medical Condition).

d) The disturbance does not occur exclusively during the course of a delirium and does not meet criteria for a dementia.

e) The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning

Specify Type :

Labile Type : If the predominant feature is affective liability.

Disinhibited Type : if the predominant feature is poor impulse control as evidenced by sexual indiscretions, etc.

Aggressive type : if the predominant feature is aggressive behaviour.

Apathetic Type : if the predominant feature is marked apathy and indifference.

Paranoid type : if the predominant feature is suspiciousness or paranoid ideation.

Other type : if the predominant feature is not one of the above, eg. personality change associated with a seizure disorder.

Combined type : if more than one feature predominates in the clinical picture.

Unspecified type

Coding Note : Include the name of the general medical condition on axis I, e.g. 310.1 Personality change due to Temporal Lobe Epilepsy, also code the general medical condition on Axis III (See Appendix G for codes).

ABBREVIATION

TBI	**:**	**Traumatic Brain Injury**
PDTBI	**:**	**Psychotic Disorder post Traumatic Brain Injury**
DSM	**:**	**Diagnostic Statistical Manual**
ICD	**:**	**International Classification of Diseases**